THE PHILIPPINE
JOURNAL OF SCIENCE
B. TROPICAL MEDICINE
VOL. XII MAY, 1917 No. 3

THE VARYING MORPHOLOGY OF BACILLUS LEPRÆ AND THE ROUTINE MICROSCOPIC EXAMINATION OF NASAL MUCUS IN LEPERS [1]

By JOHN A. JOHNSTON
(From the Biological Laboratory, Bureau of Science, Manila)

ONE PLATE

In the microscopical examination of several thousand lepers during the past three years I have been very forcibly impressed by the varying morphological appearances presented by the organism in question. In view of these observations an attempt has been made to classify the forms seen into four groups.

1. Classical type.
2. Fragmentary (degenerative) type:
    (*a*) Coarse granules.
    (*b*) Fine granules.
3. Solid type:
    (*a*) Long form.
    (*b*) Short form.
4. Nocardial or streptothrical type.

The first group corresponds to the description of the organism as usually given in the standard textbooks (Plate I, fig. *c*.). In my experience this is most frequently found in comparatively recent lesions, either of the nodular or macular type. However, it may be found occasionally in the site of old lesions which have undergone a retrogressive process, either naturally or during the administration of chaulmoogra oil, and after the period of abeyance has again become active.

The fragmentary type (Plate I, fig. *d*) is easily seen in recent lesions, although it is nearly always presented in bacteriological

---

[1] Received for publication April, 1917.

literature as the type of the organism. However, from the large number of examinations that I have made, I think that the form illustrated in Plate I, fig. *c*, is the true type. The question arises, what significance have the granules? I am not able definitely to answer this, but believe them to be indicative of a degenerative process for the reason that bacilli containing them are rarely found in active lesions, but are much more frequently seen in old inactive nodules or in the lesions of patients undergoing treatment with chaulmoogra oil.

The solid type (Plate I, figs. *e, f*) is rare, but is to be found in the discharge from an ulcer of the nasal septem or from the margins of chronic ulcers. In the case of the latter, satisfactory specimens may be obtained by the employment of Tschernogabow's technic. (6)

The fourth division is believed to be the parent form of *Bacillus lepræ* as ordinarily seen in the tissues and leproma juice (Plate I, figs. *a, o, b*). I am wholly in accord with the views of Kedrowski (2) and Bayon (1) that the organism causing leprosy has two stages in its life history: a nocardial or streptothrical nonacid-fast form and a bacillary and acid-fast one.

MICROSCOPIC EXAMINATION OF NASAL MUCUS IN LEPERS

Ever since Sticker (5) called attention to the presence of leprosy bacilli in smears made from the nasal mucosa, much stress has been laid upon this fact as a means of diagnosis in doubtful cases. Dr. Victor G. Heiser, formerly Director of the Bureau of Health of the Philippine Islands, always laid especial stress on the microscopical examination of nasal mucus in anæsthetic cases of leprosy, but he also laid stress on the fact that an ulcer or its cicatrix should be present at the junction of the bony with the cartilaginous septum. McCoy, (3) in an article published in 1915, states:

> I would especially emphasize the necessity of being cautious in drawing conclusions from the examination of nasal smears.

From my experience there is nothing to be gained from the examination of nasal mucus when definite clinical lesions are present. In the absence of clinical signs acid-fast bacilli in the nasal mucus should not be regarded as prima facie evidence of the existence of leprosy. The individual should be regarded as suspicious perhaps, and repeated examinations should be made, not omitting the examination of the circulating blood by the method of Smith and Rivas (4) during febrile paroxysm. This technic should receive far more attention than has hitherto been given to it. I believe it to be of much greater importance than the examination of the nasal mucus. The authors lay no stress on the fact that to insure success blood specimens should be taken during the febrile paroxysm.

During 1915 I took, or had taken, smears from 1,021 known lepers resident at the Culion Leper Colony and at San Lazaro Hospital, Manila, with results as shown in Tables I to III.

TABLE I.—*Examination at San Lazaro Hospital, Manila.*

Type.	Number.	Typical positive.	Typical positive.	Atypical positive.	Atypical positive.	Continued positive.
			P. cent.		*P. cent.*	*P. cent.*
Anæsthetic	37	0	0	0	0	0
Nodular	37	22	59.45	1	2.70	62.16
Mixed	95	54	56.84	3	3.15	60
Total	169	76	44.97	4	2.37	47.34

TABLE II.—*San Lazaro and Culion examinations continued.*

Type.	Number.	Typical positive.	Typical positive.	Atypical positive.	Atypical positive.	Continued positive.
			P. cent.		*P. cent.*	*P. cent.*
Anæsthetic	87	16	18.39	2	2.29	20.68
Nodular	255	131	51.37	3	1.17	52.54
Mixed	460	188	40.86	11	2.39	43.26
Total	802	335	41.77	16	1.99	43.76

TABLE III.—*Examination at Culion Leper Colony, Culion, P. I.*

Type.	Number.	Typical positive.	Typical positive.	Atypical positive.	Atypical positive.	Continued positive.
			P. cent.		*P. cent.*	*P. cent.*
Anæsthetic	124	16	12.90	2	1.61	14.51
Nodular	292	153	52.39	4	1.36	53.76
Mixed	555	242	43.60	14	2.52	46.12
Total	971	411	42.32	20	2.05	44.38

It will be readily seen from the above tables that in the anæsthetic cases where reliable findings in the nasal mucus would be of decided value only 12.9 per cent gave positive results, while 46 and 52 per cent of the mixed nodular cases gave positive findings. The conclusion may be safely drawn that examination of the nasal mucus is of no value as a routine measure.

REFERENCES

1. BAYON, H. *Brit. Med. Journ.* (1911), 2, 1269.
2. KEDROWSKI. *Zeitschr. f. Hyg. u. Infekt.* (1910), 66, 1-97.
3. McCOY, G. *Am. Journ. Trop. Dis.* (1914-15), 3, 83-97.
4. SMITH, A. J. Ibid. (1914-15), 2, 327-332.
5. STICKER. *Deutsche med. Wochenschr.* (1897), 23, 219.
6. STITT. Diagnostics and Treatment of Tropical Diseases. P. Blakiston's Son & Co., Phila. (1914), 187.

ILLUSTRATIONS

PLATE I

Types of *Bacillus lepræ*.

- *a* and *b*, nocardial or streptothrical type.
- *c*, classical type.
- *d*, fragmentary type.
- *e*, solid type, long form.
- *f*, solid type, short form.

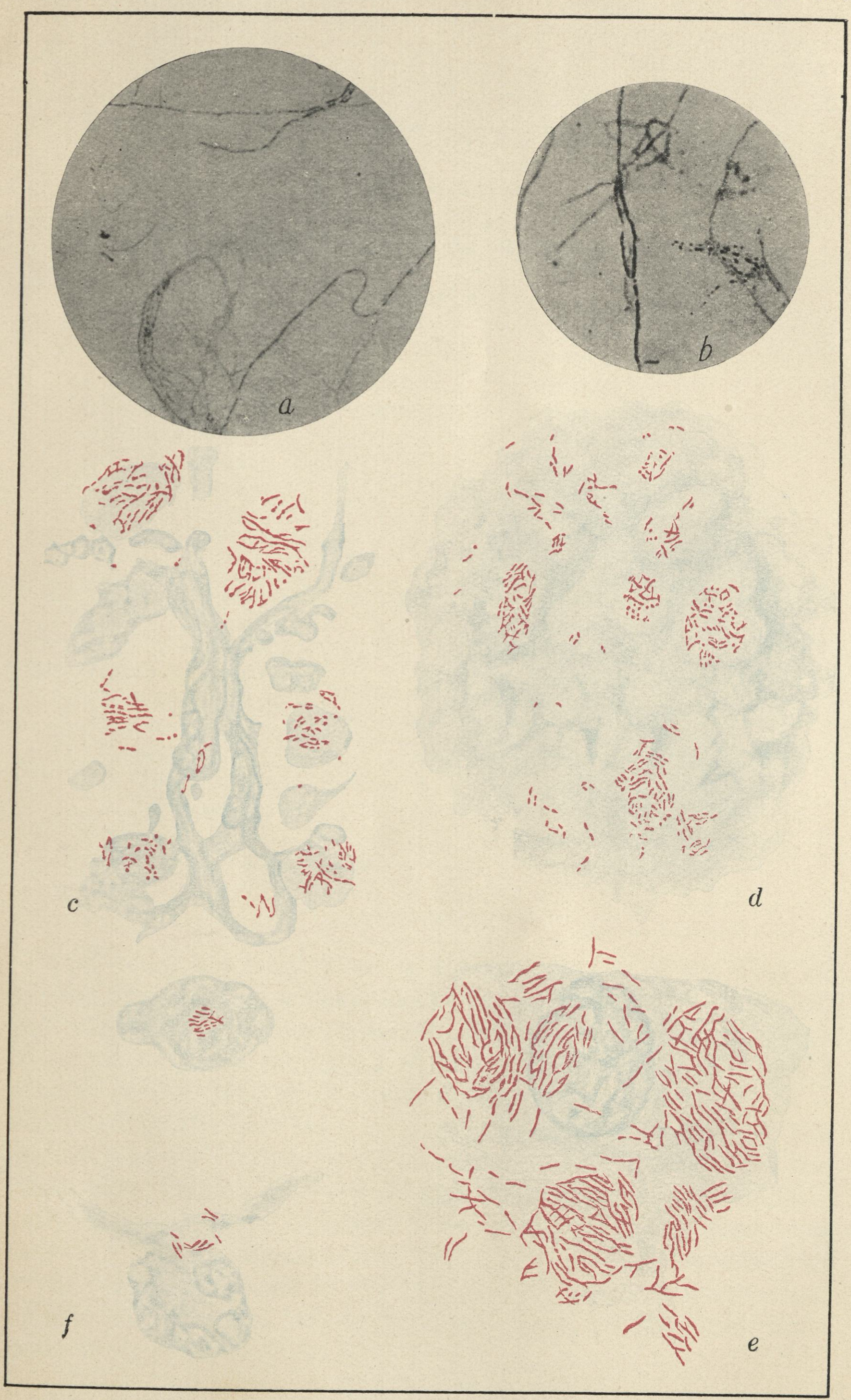

PLATE I. TYPES OF BACILLUS LEPRÆ.

AMŒBIC ABSCESS OF THE LIVER AMONG FILIPINOS [1]

By RUFINO ABRIOL

(From the Department of Surgery, College of Medicine and Surgery, University of the Philippines, and the Philippine General Hospital)

OUTLINE

- I. INTRODUCTION.
- II. ETIOLOGY.
  - 1. Geographical distribution.
  - 2. Relation to intestinal amœbiasis.
  - 3. Predisposing factors.
    - *a*. Race.
    - *b*. Malaria.
    - *c*. Vicissitudes of temperature.
    - *d*. Seasonal prevalence.
    - *e*. Personal factors.
- III. MORBID ANATOMY.
  - 1. Number of abscesses.
  - 2. Site.
  - 3. Size.
  - 4. Contents of the abscess.
  - 5. Adhesions.
- IV. SYMPTOMS.
  - 1. General considerations.
  - 2. Fever.
  - 3. Pulse.
  - 4. Rigors.
  - 5. Sweating.
  - 6. Vomiting.
  - 7. Pain.
  - 8. Cough.
  - 9. Blood picture.
  - 10. Physical signs.
- V. DIAGNOSIS.
- VI. COMPLICATIONS.
- VII. PROGNOSIS.
- VIII. TREATMENT.
  - 1. Aspiration.
  - 2. Rogers's method.
  - 3. Open operation.
    - *a*. Transpleural route.
    - *b*. Abdominal route.
  - Postoperative treatment.
- IX. CONCLUSIONS.

INTRODUCTION

Much work has been done on amœbic abscess of the liver by various observers in tropical countries as well as in subtropical and temperate localities; however, as far as I am aware, there is as yet no work presented to the medical world on amœbic abscess of the liver as it exists among the Filipinos. This is explained by the fact that, although the disease is tropical, the majority of its victims are Europeans or other peoples of the white race; hence observers on this disease are amply supplied with material from foreign patients rather than from the natives. The significance and the importance of this affection in the foreign residents in the Philippines have been emphasized by several observers, but as it exists among the Filipinos, little importance is ascribed to it.

---

[1] Submitted March, 1916, as a thesis for the degree of Doctor of Tropical Medicine. Received for publication April, 1917.

ETIOLOGY

GEOGRAPHICAL DISTRIBUTION

Tropical abscess of the liver, another name applied to amœbic abscess of the liver, as the name implies, occurs in the tropics. All authors agree that it is very common in India, Egypt, the Algerian province of Oran,(1) northern and western Africa (Gold Coast), and in Indo-China.(2) It is less prevalent in Ceylon, Malay Peninsula, Java, and Sumatra and is rare in southern China.(2) However, Manson(1) states that amœbic liver abscess along the coast of southern China is a very notable feature of the morbidity of the country. It is rare in Great Britain, and the cases met with occur most frequently in individuals who apparently had acquired the disease in the tropics. Saundby(3) reports a case of liver abscess in England, the patient never having been out of that country. In northern and central Europe it is also rare. Manson,(1) Davidson,(4) and Castellani and Chalmers(2) agree that it is relatively common in southern Europe—in Italy, Greece, the Balkan Peninsula, southern Russia, Spain, and Roumania. Reports of cases in Gibraltar, Malta, and Bengal leave some doubt as to whether or not they were contracted in these stations.(4) In Cyprus, judging from the British colonial reports, it is rare among the natives. For example, in 1903 no cases occurred among 20,000 patients treated, 261 of which were suffering from dysentery.

In Japan, although outside the tropics, it is not uncommon. The population of the southern hemisphere enjoys a practical immunity to the disease, except the Europeans in the warm northern territory of Australia and in the neighboring island of New Caledonia. Corlette(5) reports 7 cases of amœbic liver abscess occurring in New South Wales, the patients having never been outside of Australia. In the Western Hemisphere it is rare in the temperate regions, its prevalence increasing toward the tropics.(1) In the West Indies it is not as common as in India.

It is endemic in the United States. Many authors claim that the cases occurring in the United States were acquired in the tropics, or the intestinal amœbiasis of which liver abscess is a complication can be traced as originating in the warm climates. This is offset by the work or several observers. Osler informs us that it is common in the Southern States. Futcher(6) reported 119 cases of amœbiasis occurring in the Johns Hopkins Hospital, 95 of which never had been outside of Maryland. Tuttle(7) showed cases which had never been out of New York.

Boggs(8) reported 63 endemic cases in the Southern States. Among other observers corroborating the endemicity in the United States of either intestinal amœbiasis or amœbic abscess of the liver may be mentioned the names of Dock, Libman, and Spelman and Wherry.

There are no data of its occurrence in the Philippines before the American occupation, probably due to the fact that at that time it was not clinically identified and practically no studies on amœbæ had been made. Musgrave(9) reported 26 cases of amœbic hepatic abscess occurring among foreigners in Manila. Up to 1906 he never saw a case in a Filipino. Strong, McDill, Herzog, Clegg, and Fernandez,(10) however, reported a few cases in the natives. Crowell(11) reported 9 cases of amœbic abscess encountered in a series of 1,000 autopsies, 6 occurring in Filipinos and 3 in Americans. Gilman,(12) in a series of 100 autopsies, encountered it in only 1 subject.

RELATION TO INTESTINAL AMŒBIASIS

The intimate relation which exists between dysentery and liver abscess has been pointed out by many observers. Davidson(4) says:

> Liver abscess is tropical because amœbic liver abscess is confined to the tropics; and its prevalence in a given country or locality is determined by the prevalence and severity of amœbic dysentery in such a country or locality.

With older observers, namely, Prague,(13) Altschul,(13) Marston,(13) Burkardt,(13) Baly,(13) and Buchanan,(13) during whose time *Amœba* was but little known and neither bacillary nor amœbic dysenteries were clinically well identified, abscess of the liver in numerous autopsies in cases of dysenteries was hardly encountered. Showing the presence of liver abscess in cases of dysentery, Kruse and Pasquale(13) found liver abscess in 4 out of 11; Councilman and Lafleur in 6 out of 9; Strong(14) in 14 out of 96 autopsies of amœbic colitis; Osler in 23 cases out of 93 amœbic dysenteries; Robinson(15) records 12 per cent liver abscess in 96 dysentery autopsies done in the First Reserve Hospital, Manila, in 1899; Futcher(6) reports 27 liver abscesses of proved amœbic character out of 119 cases of amœbic dysentery; McDill,(16) in a review of over 100,000 cases of dysentery in the eastern countries—70,000 in India—shows 4,000 liver abscess complications. Rogers(17) found it in 20 per cent in his large series. Strong and Musgrave(17) found liver abscess twenty-three times in 100 fatal cases of amœbic dysentery; these cases were mostly American soldiers. Harris(17) found it in 15 out of 95 cases; in Craig's(18) series of 78 fatal cases 33 per cent showed liver abscess, and in his 745 collected cases of amœbic dysentery in which amœbæ were found in fæces 5 per cent showed abscess of the liver. Kartulis,(19) in 500 autopsies, showed 55 per cent amœbic abscess of the liver. Councilman and Lafleur,(19) in their autopsies, numbering 1,429, report 21 per cent with abscess of the liver. Zancarol diagnosed it in 59 per cent of 444 autopsies, Edwards and Waterman(19) in 72.1 per cent out of 699 cases, and Coffin(17) in 34 of 859 cases among American soldiers. Musgrave reports 12 cases out of 100 cases of amœbiasis in St. Paul's Hospital, Manila. McLeod,(20) in 40 cases of amœbic abscess of the liver in Shanghai, had positive evidence of dysentery in all except one, and in this there was no certainty that dysenteric lesions did not exist.

There are latent and marked infections of intestinal amœbiasis(21) which are often overlooked, the patients denying a history of dysentery and presenting no symptoms referable to amœbiasis. Some of these cases go on to recovery or death without ever showing active diarrhœa. Therefore we may have patients who have had no history of dysentery and at the same time have active amœbic lesions in the bowels. Amœbic liver abscess in such subjects gives an impression of having no relation to intestinal amœbiasis. But we must also bear in mind that there may be "individuals who had previously suffered from amœbic colitis, which had finally healed and in which the amœbæ reached the liver during the period of active ulceration of the colon."(9) This might have been a latent form of which the patient knew nothing, and autopsy in fatal cases may show only traces of previous amœbic ulcerations. Rogers(22) calls attention to the fact that amœbic lesions in the bowels may be frequently found post mortem in cases in which there were no symptoms nor history of dysentery during the treatment of the patient for liver abscess. He(22) reports 63 cases of liver abscess of which 35, or 55.5 per cent, had clinical and post-mortem evidence of dysentery; 13, or 20.6 per cent, no history, but with post-mortem evidence of dysentery; 9, or 14.3 per cent, with history, but no post-mortem evidence of dysentery; and 6, or 9.5 per cent, without a history or post-mortem evidence of dysentery. This datum gives us definite evidence of dysentery in 90.5 per cent. In another series of Rogers's(22) of 45 cases 97.8 per cent showed either the presence of active lesions or scars of former dysentery. In a third series, this time clinical cases, Rogers(22) elicited a history of symptoms of dysentery in 72 per cent. Other cases of liver abscess, showing their relation to dysentery or intestinal ulceration by observers in various countries as tabulated by Davidson,(4) appear below.

TABLE I.—*Association of liver abscess with dysentery or intestinal ulceration.*

Observer.		Number of cases of abscess.	Cases associated with dysentery or intestinal ulceration.
			Per cent.
1. Annesley	India	29	72.3
2. Waring, E.	do	204	72.2
3. Sanitary Commissioners	do	509	53.0
4. Rogers	Calcutta	63	90.48
5. Sachs	Egypt	48	41.7
6. Kartulis	do	500	55 to 60
7. Zancarol	do	444	59.0
8. Kelsch and Kiener	Algeria	500	85.0
9. Smith	Seaman's Hospital	45	84.0

On the other hand, we cannot overlook the remarks of some of the best observers in the world, who have failed to obtain evidence of a dysenteric or amœbic origin of the disease. Musgrave, in his work on amœba and amœbiasis extending for a period of many years, records cases of liver abscess "in which the most careful and persistent search failed to give evidence of a previous dysentery and there was no history of antecedent dysentery or diarrhœa." Morehead(13) failed entirely to discover any intestinal lesion in 21 of his fatal cases of liver abscesses. Kartulis(13) obtained no history, nor did he discover intestinal lesions in 33.3 per cent of his 33 cases. In 7 out of 15 cases Kruse and Pasquale(13) found no lesions in the bowels and obtained no history of dysentery; amœbæ were not identified in the contents of the abscess.

With these data before us, we are led to conclude that there are certain forms of amœbic liver abscess intimately associated with intestinal amœbiasis and that there is another form constituting a small proportion of all cases, which we may consider as a distinct disease. Musgrave(31) contends that amœbiasis of the liver through any channel other than the bowel seems improbable; however, if this be admissible, he offers three possible explanations of the apparent primary infection of the liver: (1) Amœbæ may reach the liver by the gall ducts; (2) they may penetrate the bowel wall without producing apparent lesions; or (3) the bowel lesions may have been entirely repaired.

The first of these propositions is not plausible, when we consider that amœbæ have not been found in the upper part of the intestine and in the gall bladder. However, Dr. B. C. Crowell, of the department of pathology, College of Medicine and Surgery, University of the Philippines, informs us that amœbæ have been found by him in the gall bladder. The second is tenable because Woolley and Musgrave(23) have demonstrated that amœbæ may enter the blood vessels early in the disease and may be transported to the submucosa without producing lesions. Many other observers, prominent among whom is Schaudinn,(24) corroborate this fact. In the opinion of Musgrave(23) the third furnishes the most satisfactory explanation, for it is an established fact that the process of repair, even in very extensive lesions in the intestine, is so complete that traces of the disease cannot be detected macroscopically.

Upon consulting the necropsy records of the department of pathology, College of Medicine and Surgery, University of the Philippines, of 3,630 cases, extending as far back as 1910, I find 83 cases of amœbic colitis among Filipinos, 16 of which were associated with or complicated by liver abscesses. Crowell(11) encountered 9 (29 per cent) out of 31 cases of amœbic colitis. Gilman,(12) in a series of 100 autopsies, encountered 32 cases of amœbic colitis, only 1 of which was associated with liver abscess.

In reviewing the first 42,246 cases admitted and treated at the Philippine General Hospital, I find 3,561 cases of intestinal amœbiasis, with 99 amœbic liver abscesses. Of the latter cases, 47 were discharged either cured or refusing operation, but with nothing upon which to base the diagnosis except clinical findings and, in some instances, their response to large doses of ipecac and emetine hydrochloride. Fifty-two were proved cases, of which 38 were Filipinos. Of the Filipino cases, 36 were operated upon and 16 died, 12 coming to autopsy. With the exception of 1, all showed ulcerations in the colon. However, the case which showed no lesions in the bowels had symptoms of dysentery while being treated at the hospital and showed amœbæ in the stools. Of these 12 autopsied cases, 5 gave no history of dysentery nor was there any evidence of the disease during life. In our entire series of 38 cases, 26 either gave a history of dysentery or showed evidence of intestinal amœbiasis, while in 12 cases no history of dysentery could be elicited. Four of these 12 died, showing active amœbic lesions in the colon. Therefore we have a total of 30 out of 38 cases, or 79 per cent, with either history or evidence of intestinal amœbiasis.

In regard to the relationship in point of time between the dysentery and the liver abscess, the majority of observers, namely, Musgrave,(9) Rogers,(22) Manson,(1) Davidson,(4) and many others, believe that dysentery antedates the abscess. However, there are cases on record in which the abscess precedes the dysentery. In all of our cases but one, dysentery antedated the abscess where a history or evidences of intestinal amœbiasis were obtainable. In one case the attack of dysentery started at the same time that the symptoms of abscess formation of the liver began.

With reference to the existence of amœbæ in the stools we have 12 cases in our series in which they were found; of these, 9 gave a history of dysentery and 3 denied having had it.

PREDISPOSING FACTORS

Race.—Practically all observers agree that amœbic liver abscess is more common among foreigners from a cold climate. This fact is hard to explain, especially when we take into consideration that the natives are more susceptible to intestinal amœbic infection. Manson,(1) basing his conclusions on army statistics, places the frequency of liver abscess among Europeans as compared with the natives as 95.2 to 4.8—with amœbic intestinal infection of 43.8 per thousand in the natives and 28.6 in Europeans. Hence natives are more susceptible to intestinal amœbiasis, but to the hepatic infection they are relatively immune,(25) and their immunity diminishes as they come under the influence of urban life. Of 52 cases of amœbic liver abscess, of which I have notes, 38 are Filipinos. The Chinese are much more immune than the natives of the tropics. Musgrave(9) points out that due to their excellent care of food and drink they are immune to amœbiasis. McDill(26) attributes the immunity not only to their habit of eating cooked food, but also to tea drinking.

Malaria.—Malaria is mentioned in the etiology of amœbic hepatic abscess, yet all authors of textbooks on tropical medicine agree that it has no influence in its causation. In our series, 12 had remote histories of malaria, but none of the cases had malarial infection at the time of admission to the hospital.

Vicissitudes of temperature.—Attention has been called to the comparative rarity(4) of liver abscess in countries where there is hot, moist, and equable climate as compared with other countries where the range of temperature is high and the fluctuations are sudden. Rogers(22) cites the frequency of chilling causing liver abscess, particularly in individuals who have suffered from dysentery or previous hepatic congestion of the liver. In our series, there are two cases with distinct history of chilling.

Seasonal prevalence.—Davidson(4) states that, according to the majority of the Indian authors, liver abscess is most common at the end of the rains and at the beginning of the cold season. Rogers,(27) however, in comparatively recent work on seasonal prevalence of amœbic colitis, concludes that the most frequent occurrence of this intestinal infection is at the beginning of the rainy season. Deeks and Shaw(28) have the same to say in Panama with regard to the prevalence of amœbic colitis. If we take the prevalence of amœbic dysentery as a basis for the occurrence of amœbic liver abscess, we would naturally think that liver abscess would also be more prevalent at the beginning of the rainy season rather than at the end. Table II shows the distribution of our cases by months considered at the onset of the symptoms referable to liver abscess. In the first instance we notice the predominance of the cases during the cool months, that is January and February. With regard to time of onset, we have the majority of the cases during the rainy season.

TABLE II.—*Distribution by months of 38 cases of liver abscess among Filipinos.*

Month.	On admission.	At time of onset of abscesses.
January	4	5
February	7	4
March	1	0
April	1	4
May	3	1
June	3	2
July	2	4
August	3	5
September	5	6
October	5	1
November	3	2
December	1	4
Total	38	38

Dividing our cases into groups representing the admission during periods of three months, or quarterly, we find the same results as obtained in the above table. In the first quarter, the cooler part of the year, we have the majority of the cases at the time of admission and at the time of onset. The predominance is confined to the third quarter, that part of the year with the most rainfall.

TABLE III.—*Quarterly distribution of liver abscess among Filipinos.*

Quarter of year.	On admission.	At time of onset of abscess.
First	12	9
Second	7	7
Third	10	15
Fourth	9	7

Personal factors.—Liver abscess may occur at any age. Our youngest case was 9 years old and the other extreme was 69 years. It is commonest between the ages of 21 and 40. Among our 38 cases, 22 occurred between these ages. The average age was 36 years and 9 months. Rogers,(22) in a review of 300 cases of liver abscess among Indians and 92 among Europeans, comes to the conclusion that the age incidence among Indians and Europeans is similar. The majority of his cases both in Europeans and Indians was between the ages of 21 and 40. Reports on cases in old age are scanty. Gilman, in 1914 at the December meeting of the Manila Medical Society, reported a case of liver abscess in an Englishman who was 83 years old. Amberg(17) reports 5 cases in children, and Rolleston(13) collected 16 similar cases.

In all endemic regions there is a predominance of cases in men over women. Davidson(4) estimates the proportion of cases in men to women as 30 to 1. The Indian statistics(4) for 1901–3 give the ratio of 7 to 1. Futcher(6) had a ratio of 8 to 1 in his Johns Hopkins series. Many observers claim that women are relatively immune to the disease, because they are less addicted to alcohol and less exposed to other exciting causes of the disease. "Van der Burg(13) ascribes it to menstruation, and this may have a certain influence in diminishing liability to hepatic conditions and indirectly to abscess." If the first argument be tenable, and considering that the Filipino woman of the lower class is as much subject to intemperance and as exposed to other exciting causes of the disease as the man, we may with justice ascribe to this the relative frequency of amœbic liver abscess among Filipino women. In our series, seven occurred in women. This gives us 22.8 per cent of all the cases, and the ratio of the female cases to males is 7:31 or 1:4.43 +.

In the opinion of most observers alcohol, particularly in excess, plays an important rôle in the causation of the abscess, and here we may say that the relative frequency of the disease in Europeans and Americans is ascribed to the fact that they are more addicted to liquors than the natives. Likewise European women and children are less intemperate, and hence the infrequency of the disease in these individuals. Musgrave(9) warns us of giving alcohol a too prominent place in the etiology of liver abscess, basing his conclusions on his series in which alcohol was not an etiological factor. In our series, four gave a history of drinking "vino," [2] but as all of these except one had a recent history of dysentery, we cannot conclude that alcohol was an etiological factor in these instances.

Excess of animal food and use of hot condiments such as curries and probably pepper are said to contribute, along with other causes, in the formation of liver abscess. Davidson(4) states that the influence of excesses of this kind is overestimated, and cites the infrequency of liver abscess among the better class of natives of India who are especially addicted to the vice. All our cases, except one, belong to the middle and lower classes, and we can safely take for granted that gluttony is a rarity, if not absent, among these people.

While in the standard textbooks on tropical diseases no mention is made of the rôle of occupation in the causation of liver abscess, it is of interest to note that 11 of our cases occurred among laborers, 7 among clerks, 4 farmers, 3 soldiers and policemen, 3 housekeepers, 2 fishermen, 2 laundresses, one each in a driver, lawyer, and musician, one in a child, and two without occupation. In connection with amœbic colitis, Deeks and Shaw(28) point out that occupation is not an etiological factor, except as it may bring the individual to a position or condition where infected water is solely available for his consumption. In view of this exposure to infection because of occupation, is it not justified to assume that occupation is a factor? Farmers, fishermen, and laborers are the class of Filipinos nearest to nature, and the kind of water available for their consumption is rarely free from amœba. As already stated, 17 of our cases occurred among these classes of people.

MORBID ANATOMY

NUMBER OF ABSCESSES

The old observers always regarded amœbic abscess of the liver as occurring singly. There is not the slightest doubt that amœbic abscess of the liver may occur in multiple numbers. Craig(18) describes cases in which the abscesses could be counted only with great difficulty. Reading the results of various observers, we note that there is a discrepancy in results. Waring,(13) in an analysis of 288 cases, found that 61.5 per cent were single; Niblock,(13) 83 per cent; Rouis,(13) 75 per cent; the Sanitary Commissioner for India in 509 autopsies in 1896–1901 found that only 34 per cent were single. Craig,(18) in an analysis of 24 cases, encountered only 10 single and 14 multiple. In our cases, 6 were multiple and the rest single, the latter giving a percentage of 84.

---

[2] An alcoholic beverage with about 19 per cent alcohol.

SITE

The majority of our cases are in the right lobe, the upper and lower portions being the favorite seat. Thirty-four cases involved the right lobe. Of these, in 2 the left lobe was also involved and in 1 case the abscesses were distributed throughout all lobes. Two cases were confined in the left lobe. Of those occurring in the right lobe, 10 were in the superior aspect and 11 in the lower part and 13 cases had no definite data as to what part of the right lobe was involved. Rouis,(20) in an analysis of 639 cases, gives the occurrence in the right lobe as 70.85 per cent; in the left lobe as 13.3 per cent; and in the lobus Spigelii as 0.3 per cent. Craig,(28) out of 24 cases, encountered 19 in the right lobe, 8 in the left lobe, and 1 in the lobus Spigelii.

SIZE

When we bear in mind that some cases of amœbic abscess of the liver may exist in a person, particularly among the natives, without uncomfortable symptoms, the size of the abscess when it comes to the surgeon's hands must of necessity vary. The abscesses range from 1 millimeter(28) in diameter to one case containing as much as 3,500 cubic centimeters of pus. When multiple, they are generally small. Often two or more abscesses form a single one by confluence. In several of our cases, the abscesses were large enough to yield 2,000 cubic centimeters of pus each.

CONTENTS OF THE ABSCESS

The characteristic amœbic pus is diagnostic in itself. Practically in all our cases the pus is viscid and brown in color, resembling thick chocolate. It is odorless and is streaked with blood. The blood may be fresh, being a contamination from the operative incision, or may consist of old clots. Occasionally we may find lumps of translucent mucoid yellowish material which as a rule harbors encysted amœbæ. Microscopically the pus is rich in necrotic liver tissue. Leucocytes, red blood cells, and Charcot-Leyden crystals are also found. Unless there exists a secondary infection, the pus is bacteriologically sterile. With the presence of bacteria, the color and consistency differ entirely.

In 25 cases in our series the pus was examined and amœbæ were identified in 9 cases. *Bacillus subtilis* was present in one case, staphylococci in another; in a third case colon bacillus was present as well as *Oxyuris vermicularis.* Our findings in pure amœbic cases as to the character of the pus correspond very well with the observations of workers in other countries.

ADHESIONS

These occur in cases where the abscess is near the surface of the liver. We had one case in which there were so many adhesions around the abscess that during an attempt to separate them the abscess ruptured. In another case the abscess was on the anterior superior surface of the liver, and there were many recently formed adhesions around it, the abscess having ruptured before operation. In spite of the adhesions in this particular case, there was marked secondary peritoneal infection.

SYMPTOMS

GENERAL CONSIDERATIONS

All observers agree that there are no fixed symptoms which make up a classical clinical picture of amœbic liver abscess. It is true enough that there are certain cases which we can correctly diagnose before operation, but the range of variation of the many clinical signs is so great that experience has compelled us to be more conservative in ascribing the symptoms to liver abscess only. Certain abscesses give typical symptoms of fever, chills, pain, and enlargement of the liver and yet many cases of the large variety show no symptoms whatsoever.

FEVER

The fever, just as the symptoms, varies in degree. Four of our cases had perfectly normal temperature; 6 cases had fever ranging from 37° to 38° C. at the time of admission, but twenty-four hours after the patients had been put to bed and proper dieting instituted, the temperature came down to normal. Twelve cases presented an irregular temperature curve, rarely reaching, however, 39.5° C.; 7, with temperature of the remittent type with normal or slightly subnormal in the morning and 37° to 38° C. in the evening; 2 cases with septic temperature; 2 cases with subnormal, 1 remittent and 1 with a continued fever between 37° and 38° C. until twenty-four hours after operation. Musgrave(9) states that there is no characteristic fever in liver abscess; Rogers(22) places the type of fever as remittent in over half of the cases; one seventh, high continued, keeping above 101° F. and varying more than two degrees in the twenty-four hours; in one fifth, the temperature was intermittent; and in 2 out of 57 cases it was normal. While all authors agree that some liver abscesses of the amœbic type at times present no fever, data as to the percentage of the cases in which this exists is not forthcoming except in Rogers's cases cited above. Our Filipino cases of 4 with normal temperature out of 38 cases probably represent the true proportion among the Filipinos.

PULSE

The pulse varies according to the degree of temperature present, increasing directly in proportion with the fever.

RIGORS

In older descriptions of amœbic liver abscess, rigor is given much prominence. Eleven of our cases gave a distinct history of rigor. This, as a rule, occurred in our cases toward evening as a mild chilly sensation, never so severe as that found in malaria. Slight fever follows it.

SWEATING

Rogers(22) and Manson(1) emphasize the presence of copious sweats and state that it may be so abundant as literally to drench the patient's clothes. This particularly occurs when the patient sleeps. In our series four had sweats to a moderate degree, three of the cases were among those who had rigors, and the perspiration occurred immediately after the chill.

VOMITING

In regard to vomiting, very little attention is given to it by many observers. Manson(1) attributes it to pressure on the stomach or a catarrhal condition of that organ. In our cases twelve gave symptoms either of nausea or vomiting.

PAIN

As to the typical description of the pain referable to liver abscess, Manson(1) says:

> There are several types of pain—local and sympathetic—associated with liver abscess. Complaint is almost invariably made of a sense of fulness and of a sense of weight in the region of the liver, not unfrequently referred to the infrascapular region. Stabbing, stitch-like pain, increased by pressure and especially by deep inspiration, by coughing and all sudden jarring movements, is very common, and probably indicates perihepatitis from proximity of the abscess to the surface of the organ. Percussion, or firm palpation, especially if practised during deep inspiration and below

the ribs in front, generally causes smart pain and decided shrinking, the rectus muscle starting up as if to protect the subjacent inflamed parts.

In our series 34 had symptoms of pain in the right hypochondrium; in 1 case in which there was encountered rupture of the abscess into the peritoneal sac, at the time of the operation, the pain was generalized in the abdominal region. In another case in which the location of the abscess was in the right posterior side of the right lobe the pain was in the right side of the chest and in the right shoulder. In a third case the pain was localized in the right iliac region. In this the cæcum was the seat of extensive amœbic lesion; there were multiple abscesses in the right and left lobes, one of the latter having ruptured and formed a subphrenic abscess in the left hypochondrium. In a fourth case the epigastrium and left hypochondrium were the seat of the pain. The abscess in this case ruptured and formed a subphrenic abscess. In one case the onset of the disease started as aching of the long bones. This same patient had his pain in the hepatic region aggravated by ingestion of food.

One frequent symptom in our cases, particularly among those well advanced, was a peculiar sense of fulness in the right upper quadrant of the abdomen. In cases of this kind, as a rule, the patients came to the hospital for no other complaint but this, and they themselves were aware of the presence of a swelling in the upper part of the right side of the abdomen.

COUGH

Even in cases uncomplicated by rupture of the abscess into the lungs or actual involvement of the pleura through a process of continuity, cough plays an important rôle in the symptomatology. In abscess of the superior surface of the liver, the irritation on the diaphragm excites a dry, hacking cough. Even in those located in the lower segment of the right lobe, the distension upward may be sufficient to cause coughing. Eight of our cases gave this symptom, three of whom had the abscess in the lower part of the right lobe.

BLOOD PICTURE

In cases observed by Manson,(1) Rogers,(22) and Musgrave,(9) no typical blood picture was found. Rogers(22) mentions as high a count as 35,000 and 38,000 leucocytes in two cases, both of the multiple variety. Our case with a count between 25,000 and 30,000 leucocytes was also in a multiple type. Musgrave(9) attributes high leucocytosis to intestinal infection. Table IV shows the leucocytosis in our series.

TABLE IV.—*Leucocytosis in liver abscess among Filipinos.*

Number.	Cases recovered.	Fatal cases.	Total.
6,000-10,000	4	4	8
10,001-15,000	9	4	13
15,001-20,000	4	5	9
20,001-25,000	2	1	3
25,001-30,000	1	0	1
(a)	2	2	4
Total	22	16	38

[a] Count not made.

We note in this table that eight cases had a count between 6,000 and 10,000, which is practically normal.

Rogers(22) has pointed out that in liver abscess the increase in the polynuclears is less marked than in cases of septic infection.

Table V shows the variation in polynuclear counts in our cases.

TABLE V.—*Polymorphonuclear leucocytosis in liver abscess among Filipinos.*

Per cent.	Cases recovered.	Fatal cases.	Total.
Less than 60	1	0	1
60-65	1	0	1
66-70	1	0	1
71-75	5	1	6
76-80	4	1	5
81-85	2	5	7
86-90	4	3	7
91-95	0	2	2
(a)	4	4	8
Total	22	16	38

[a] Count not made.

The two cases with counts above 91 per cent may be accounted for in one instance by a colon bacillus infection and in the second by the presence of an independent empyema in the lungs, not secondary to rupture of the abscess. Chalmers and Archibald,(29) in their recent work on the importance of differential counts in uncomplicated intestinal amœbiasis, call attention to the presence of eosinophilia without the presence of worms. The same authors emphasize the mononuclear leucocytosis especially in latent cases as means for diagnosis. If this be the case with uncomplicated cases of intestinal amœbiasis, can we not apply the same principle in hepatic amœbiasis of uncomplicated variety? However, in our series there is only one case which had an eosinophilic count more than normal, and this showed in the stools the eggs of *Trichocephalus dispar, Ascaris lumbricoides,* and *Ankylostoma.* Table VI shows the mononuclear leucocytosis:

TABLE VI.—*Mononuclear leucocytosis in liver abscess among Filipinos.*

Per cent.	Cases recovered.	Fatal cases.	Total.
0-1	13	8	21
2-5	3	2	5
6-10	1	2	3
11-15	0	0	0
16-20	1	0	1
([a])	4	4	8
Total	22	16	38

[a] Count not made.

PHYSICAL SIGNS

The majority of our cases were cachectic, but some of them looked perfectly healthy and well nourished. Marked jaundice was never observed, but a slight yellowish tinge in the conjunctiva was noticed in eighteen cases. Those cases too sick to be about generally lay in bed on the right side with the lower limbs drawn up.

In two of our cases ascites was present, and three had œdema of the legs without nephritis. Two cases presented symptons of hemorrhoids, and in one case the superficial veins of the anterior abdominal wall were enlarged.

Respiration was proportionate to the fever, if any was present. In those cases with marked pain in the hepatic region, the respiratory movements were limited to the thorax. Auscultation revealed râles or pleuritic rub at the base of the right lung in four cases.

The hepatic dullness varied greatly. In our cases in which abscess was located in the upper segment it reached as high as the fourth rib in the midclavicular line; the lower border was not changed at all in some cases. In abscess of the lower segment of the right lobe the liver becomes prominent in the right hypochondrium. In 27 of our cases there was a definite tumor palpable below the right costal arch. In 2 cases fluctuation was distinct. Tenderness was on the whole very slight except in those cases with mixed infection, and if a moderate or marked degree of abdominal rigidity is present, it is localized in the upper segment of the right rectus.

DIAGNOSIS

There are certain cases of amœbic liver abscess whose symptoms are so prominent that one can readily make a correct diagnosis. I have already stated that the symptoms of this disease are varied and due to this we are not always in a position to give a correct diagnosis in certain cases. Manson (1) puts it thus:

> Golden rules in tropical practice are to think of hepatic abscess in all cases of progressive deterioration of health; and to suspect liver abscess in all obscure abdominal cases associated with evening rise of temperature, and this particularly if there be enlargement of or pain in the liver, leucocytosis and a history of dysentery—not necessarily recent dysentery. If doubt exist, there should be no hesitation in having early recourse to the aspirator to clear up diagnosis.

Chief among those diseases which may be confused with liver abscess are syphilitic disease of the liver, cholecystitis and suppurative cholangitis, malarial hepatitis, carcinoma of the liver, pyelonephrosis or hydronephrosis, hydatid cysts, and tuberculous abscess of the abdominal wall. Syphilitic disease of the liver may be excluded by the Wassermann test and therapeutic tests with salvarsan and other antisyphilitic agents. Cholecystitis and suppurative cholangitis are distinguished by high leucocytosis, and in the latter there is apt to be present a distinct septic temperature. The liver, as a rule, is not changed in size. In some of our cases the exclusion of these conditions has not been made before laparatomy was performed. Malarial hepatitis is easily ruled out by the presence of the malarial parasites in the blood and the quick response of the disease to quinine. Carcinoma of the liver has often led us to a mistaken diagnosis. Careful examination shows an enlarged liver of a firm consistency, and sometimes nodules are distinctly palpable. Pyelonephrosis or hydronephrosis, especially when the colon has become adherent to the liver, complicates the diagnosis. Catheterization of the ureters will as a rule lead to their identification. Hydatid cysts are slow in their growth, painless, and devoid of constitutional symptoms. Exploratory puncture may be resorted to in order to observe the character of the fluid in the cavity. Tuberculous abscess of the abdominal wall often leads to a doubtful diagnosis. In some large abscesses of the lower part of the right lobe, particularly when fluctuation is present and when complicated by adhesions of the liver to the parietal peritoneum in a subject with thin abdominal wall, the differentiation is hard to make. Occasionally an abscess of this type does not move with respiration, and this may be considered a differentiating point. However, exploratory puncture is easily done and settles the diagnosis.

In abscess occurring in the upper segment of the right lobe of the liver, it is natural to assume that the bulging will take place upward, particularly if the location is near the capsule. This, no doubt, explains the relative frequency of the enlargement of the dome as claimed by Osler.(30) The upward enlargement is often hard, if not impossible, to determine by simple percussion. In such cases as these radiography assists very much in ascertaining whether or not there exists an abscess. Of course, any disease that gives rise to an upward enlargement of the liver will lead the radiologist to suspect abscess of the liver; hence the necessity of an experienced eye in interpreting the shadows of the dome of the liver. Fernandez(10) states that abscess in the superior surface of the liver causes a difference in the level of the left and right sides of the diaphragm of 6 to 8 centimeters. Twelve of our cases have been referred to the radiologist for confirmation of the diagnosis. In nine cases a positive diagnosis of liver abscess was reported; in one case, it was probable abscess; in another case, only a statement of enlargement of the liver downward was reported; and in a third case the findings were normal. Among the positive cases, one was of the multiple variety involving the right lobe; three were abscesses of the lower part of the right lobe of fairly large size, probably sufficiently extensive to involve the upper part of the lobe also; and in four cases the lesion was in the dome. The abscess in the doubtful case was in the lower part of the right lobe; the case with a report of enlargement downward presented an abscess in the lower part also; and lastly, the case with normal liver had an abscess in the left lobe.

After analyzing the above results of radioscopic examination of liver abscesses, we cannot accept without question the unfailing value of X-rays as an aid to the diagnosis of liver abscess. However, there is absolutely no doubt that a radiograph, considered in correlation with other symptoms pointing to hepatic abscess, is of great value.

COMPLICATIONS

Aside from the colon lesions practically all complications of the liver abscess arise from its spontaneous rupture. Complications which may thus be produced are numerous, their character depending upon the viscera or cavities involved. When the contents of the abscess rupture into the abdominal cavity, a typical acute peritonitis is produced. Dudley(31) reports a case of this kind with operation and recovery. Rupture into the pleural cavities or pericardium, into the lungs, into the stomach or colon, into the right kidney, and into the portal vessels may take place, giving rise to symptoms referable to the particular structure involved. Cyr(32) gives the results of various observers as follows: Rupture into lung, 59; pleura, 31; pericardium, 1; peritoneum, 39; stomach, 8; intestines, 13; kidney, 2; inferior vena cava, 3; bile passages, 4; externally, 2. Craig,(18) in 7 cases, reported 2 ruptures into the pericardium and 5 into the pleura. In our series 6 of 12 fatal cases had ruptured spontaneously, 4 of these into the peritoneum and 1 into the pleura.

Another peculiar complication of liver abscess is intestinal hemorrhage, ending fatally as a rule. Reports on this complication occur in cases with active lesions in the colon. Strong(33) reports four cases which terminated fatally. Two of our cases presented this complication, one without operation, which ended fatally. In the other case a hemorrhage in the abscess cavity occurred one month after operation, and this was followed twenty-four hours later by hemorrhage from the intestines, about 200 cubic centimeters of bright red blood having been passed per rectum. Elevation of the lower part of the bed, packing the abscess cavity with gauze, administration of emetine hydrochloride intravenously, and subcutaneous injection of normal horse serum saved the patient.

PROGNOSIS

Amœbic liver abscess runs a course of varying length. Manson(1) says that generally it is an affair of several months. If proper treatment is instituted early, its course is greatly shortened. If it ruptures, the termination varies with the nature of the visceral involvement. Ruptures occurring into the lungs, stomach, and intestines usually result favorably. At times, small abscesses become encysted or absorbed and thus end in recovery. The coexistence of intestinal amœbiasis, intestinal hemorrhage, broken down state of constitution, and multiplicity of the abscesses add materially to the gravity of the disease. Likewise the existence of mixed infection renders the outlook less favorable.

The death rate among Europeans is high, but is comparatively low among those who come early under observation. Table VII shows a comparison of the percentage of mortality as given by various observers.

TABLE VII.—*Mortality in amœbic liver abscess by various observers.*

Observer.	Cases.	Country.	Mortality.
			Per cent.
Rouis (1)	203	Algiers	80
Castro (1)	125	Egypt	72.5
Rogers (22)	64	India	53
Megaw (22)	292	India	60.1
Rogers (22)	52	India	73
Abriol	38	Philippines	31.5

We note that the mortality among the 38 cases of Filipinos is relatively low, in spite of the fact that most of our cases come under observation at a very late stage of the disease and many of them are not by any means in a favorable condition for surgical intervention. It is hoped that with the growing popularity of operative procedures among the natives we will be able to reduce the death rate to a very low minimum.

TREATMENT

When the diagnosis of amœbic hepatic abscess is definitely established, the treatment resolves itself into various methods of surgical procedure. These range from a simple aspiration and injection with quinine to freely opening and draining the abscess.

ASPIRATION AND INJECTION OF QUININE WITHOUT DRAINAGE

Rogers in 1902 was the first to suggest aspiration and injection of quinine without drainage. Later Wilson,(34) Spencer,(34) O'Kenealy,(34) and Stevens(34) reported favorable results from this procedure. Rogers(22) describes the technic as follows:

> In carrying out this plan of treatment the following points require attention. The skin at the seat of puncture must be most thoroughly sterilized to prevent any bacteria being carried into the cavity. If the presence and position of the abscess are accurately known a full sized aspiration trocar should be used to allow as much as possible of the thick pus being withdrawn through it. For the same reason it is also an advantage to use a T tube of large calibre fitting into the exhausted bottle. Messrs. Down Bros. have made a suitable one for me. The cavity is emptied as far as possible, some of the first pus being run directly into a sterile test tube for bacteriological examination. A previously boiled solution of the very soluble bihydrochlorate of quinine, of a strength of 10 grains in 1 oz. of water, is now injected into the abscess cavity through the cannula by means of a sterile syringe, and the cannula is then withdrawn

and collodion applied externally. If only a few ounces of pus are obtained, it will be sufficient to inject 2 oz. of the quinine solution, but if a pint or more is present then 4 oz. containing 40 grains of quinine should be used, so as to saturate the whole wall. In some cases the temperature falls finally, all the symptoms disappear and weight is rapidly gained after a single injection, as happened in eight cases. More frequently the effect is only temporary, and it is then advisable to repeat the little operation after about a week, when less pus is commonly obtained. A third injection is not rarely required, but in large abscesses even more, perhaps even four or five, may be required. I have noticed that if an originally present leucocytosis completely disappears, little or no pus is usually obtained at a second aspiration, and uninterrupted convalescence ensues. The continued presence of even a slight leucocytosis is generally an indication for repeating the aspiration. In the common fibrous-walled single abscess no cinchonism results, even from the injection of as much as sixty grains of quinine, but in more acute ones the drug may be absorbed to some extent into the circulation.

ROGERS'S FLEXIBLE SHEATHED TROCAR FOR STERILE SIPHON DRAINAGE AND QUININE IRRIGATION

With a special apparatus Rogers drains aseptically the cavity of the abscess and washes it out with sterile quinine solution to kill the amœbæ.

OPEN OPERATIONS WITH DRAINAGE

There are various ways of approaching the liver with a view of performing a simply oncotomy. In the main they are divided into two types: the transpleural route and the abdominal route. Some surgeons prefer to explore the liver to determine the presence and location of the abscess before proceeding to operate; others expose the liver and use the needle to locate or determine the presence of an abscess.

Transpleural route.—Several surgeons describe various technics of the transpleural route; all have their advantages and objectionable points. McDill(16) describes a technic in which pneumothorax is obviated. Essentially it is as follow: A curvilinear incision to expose the right ninth and tenth ribs is made in the midaxillary line. All the tissues down to, but not including, the fascia overlying the muscles are reflected in one flap. The periosteum of the exposed ninth rib is dissected out and 10 centimeters of it is resected. The resulting gutter is closed with a running catgut suture through the entire thickness of the musculature. Just above the tenth rib an incision is made through all layers of the chest wall and the diaphragm. While this incision is accomplished, the fingers of an assistant firmly press the loosened chest wall inward and against the diaphragm to prevent pneumothorax when the incision opens or crosses the pleural sac. The diaphragm and the chest wall are finally sutured and the liver explored with a needle. If abscess is found, it is either incised or left alone for forty-eight hours, allowing parietovisceral adhesions to form, before incision is made. A short rubber tube is used for drainage.

Abdominal route.—While all the operations of this type are essentially the same in principle, there are important differences in the technic either for attaining better asepsis or better drainage.

The principle of Manson's method is briefly as follows: A trocar and cannula 9 millimeters in diameter and 10 centimeters long is used. Through this a rubber drainage tube stretched by a long stilette, the extremities of which are placed in buttons tied into the ends of the tubing, is introduced. The trocar and cannula are thrust into the liver, and the tubing is stretched by the stillete introduced inside the cannula after removal of the trocar. The cannula and the stillete are then removed, allowing the rubber tubing to contract and fit snugly into the punctured wound in the liver, thus preventing the escape of pus and thereby contaminating the serous sac and at the same time lessening the chances of hemorrhage. Cantlie(34) reports 82 per cent recoveries in 100 cases by operation of this method.

Rhoads,(35) realizing the possible dangers associated with aspiration as a method of locating the abscess, and the uncertainty of obtaining reliable information, advocates direct palpation of the liver through a high, right rectus incision in every suspected case, as a more satisfactory method for locating the pus collection. If the abscess is located in the lower part of the right lobe, lobus Spigelii, lobus quadratus, or the left lobe, evacuation is accomplished as follows according to the words of the author:

> The assistant being ready with gauze sponges clamped to carriers, an incision 4 cm. in length and 1 cm. or more in depth is made through the capsule. If this has not reached the pus, a closed broad-ligament clamp is pushed through the opening, past the fibrous boundary of the abscess and into the cavity, opened so that the blades are separated at least the length of the surface cut, and withdrawn. The fibrous wall is very dense in some cases, varying from .5 cm. to 2 cm. in thickness, according to the age of the abscess, and may require considerable force to overcome its resistance as the clamp is pushed through. As the thick, brownish-yellow pus appears along the path of the instrument, the assistant sponges it away rapidly from the opening, as little of the material as possible being permitted to flow over the gauze protecting the intestines. The clamp is inserted on successive times as the flow lessens until the cavity has emptied itself, and its

interior is then examined with the finger. If trabeculae divide the cavity into various compartments, they are broken down, and loose shreds of necrotic tissue hanging from the shaggy wall are brought away. The interior is wiped dry with narrow strips of gauze armed on a clamp, several of the strips being allowed to remain in the cavity after it is comparatively cleansed, and the free ends are brought to the surface of the abdomen to act as a drain. Alongside these strips is inserted a large, firm, rubber tube, 1.5 cm. in diameter, with 2 openings cut into its sides near the end projecting into the abscess cavity, and the free end is anchored in the abdominal incision at the point best adapted for drainage. The soiled gauze strips protecting the intestines are removed after clean strips in equal number have been placed underneath them, i. e., between the original gauze strips and intestines, and these in turn are substituted by one strip, 10 cm. wide, for abdominal drainage, carried to the farthermost point posteriorly underneath the liver, and so placed that when spread out its edges will overlap the liver incision. If the operation was on the upper anterior surface of the liver the gauze protecting the suprahepatic space is removed, the space is wiped dry, and narrow gauze strips 4 cm. wide are placed on each side of the liver cut and brought out of the abdominal wound. We have then these gauze strips for drainage through the abdominal wall, beside the rubber tube; 1 piece 10 cm. wide, and of 8 thicknesses, for the abdomen; 2 narrow strips for the abscess cavity (2 narrow strips being used in preference to 1 broader one on account of ease in withdrawal); and if the abscess was on the upper anterior surface, 2 additional narrow strips as guards of the suprahepatic space, 1 on either side of the liver cut. The destination of each set of strips is marked by tying black or white sterile silk to the free ends before the abdomen is closed.

Dr. P. K. Gilman, of the department of surgery, University of the Philippines, in his wide experience with amœbic hepatic abscess has evolved a very good operation. In this method the high, right rectus incision is utilized and a thorough digital examination of the liver is made. In certain cases where this method fails, a narrow gauze packing such as that used in uterine operations is tucked in around a circumscribed area of the liver, walling off the abdominal cavity. A long aspirating needle attached to a glass syringe is thrust, in various directions, into the liver substance. Once the abscess cavity is discovered, the needle is withdrawn and a large Ochsner's gall bladder trocar attached to a long rubber tubing is pushed into the abscess cavity. As a rule the thick viscid liver pus flows out freely, but if the flow is sluggish, a large syringe may be used to suck the pus out. With a little pressure exerted upon the abdomen to cause the intestines to push up against the inferior surface of the liver, the abscess cavity may be completely evacuated; however, complete evacuation is not necessary. Immediately after withdrawing the trocar, one end of a piece of rubber tubing, slightly larger than the trocar and 60 to 70 centimeters long, is introduced into the abscess cavity by means of an artery clamp. Pieces of one-inch iodoform gauze are placed on the liver surface immediately around the rubber tube and covered by the lips of the incision when the latter is finally sutured. After removing the packing used for walling off the other viscera, the incision is closed in layers, anchoring the tube to the edges of the incision with silkworm gut sutures. To the ends of the iodoform gauze safety pins are fastened to prevent the chance of losing them. When the patient is returned to his bed, the distal end of the rubber tube is placed in a large white bottle suspended from the middle portion of the right side of the bed.

This method has two very good features which make it the operation of choice. These are the use of Ochsner's gall bladder trocar, which obviates the chances of contaminating the peritoneal cavity by the avoidance of the overflow of pus in the operative field, and the postoperative drainage by means of the long rubber tubing. With our cases we have observed that during operation the abscess cavity does not contract very much, a fact which is especially true with old abscesses with relatively thick walls. However, during the three or four days following operation, much contraction takes place. In some cases this was apparent from the accumulation of as much as a liter or more of pus in the drainage bottle during the first twelve hours following operation. As the pus passes through the tube easily, we derive another advantage of keeping the operative wound and the dressings perfectly clean.

POSTOPERATIVE TREATMENT

In the open operations described above all the drainage tubes provided are just long enough to protrude out of the incision. Fairly thick dressing is applied over the wound. This is changed as often as demanded by the amount of discharge. If the tube is acting properly, it is not disturbed during the first week. Later it may be removed and cleaned, and as the discharge diminishes in amount, it may be gradually shortened. In the majority of our cases the abscess cavity was not irrigated with any kind of solution, while in a few others in which the discharge showed amœbæ quinine in 1 to 1,000 solution and fluid extract of ipecacuanha in a solution of 1 to 500 were used. In cases with intercurrent intestinal amœbiasis, emetine given intravenously, 1 grain daily, and ipecacuanha, in salol-coated pills, were administered. The value of emetine hydrochloride in extremely ill cases cannot be overestimated, for we have saved many such by administration of this drug. The after treatment in cases operated upon by Gilman's method is essentially the same. After one week the tube is cut near the operative incision and covered with dressings.

CONCLUSIONS

1. Seventy-nine per cent of liver abscess cases among Filipinos as seen at the Philippine General Hospital are distinctly associated with intestinal amœbiasis.
2. Liver abscess is relatively frequent among Filipinos.
3. There is apparent predominance of cases of liver abscess among Filipinos during the cool and rainy months.
4. The age incidence corresponds with that of other countries.
5. Filipino women are not as immune to the disease as European or American women.
6. Occupation plays an important rôle in the etiology of liver abscess, as in certain occupations the individuals are brought to a condition or position favorable to infection.
7. The right lobe is the most frequent site of the abscess in our cases among Filipinos.
8. Fever is not typical in cases of amœbic abscess of the liver.
9. There is nothing characteristic in the pain symptoms.
10. The blood picture varies—being normal in many cases. In the majority of the cases, the polynuclear count is normal. There is no eosinophilia in our cases, nor is there any increase in the mononuclears.
11. Diagnosis is easy in advanced cases, but extremely difficult in early cases with small abscesses.
12. Intestinal hemorrhage as a complication of liver abscess is not necessarily fatal.
13. Mortality among Filipino cases is relatively low, in spite of the fact that they come very late for treatment.

In conclusion, I desire to express my gratitude to Drs. W. E. Musgrave, P. K. Gilman, and B. C. Crowell and the Misses Esperanza Concepcion, Carmen Ocampo, and Catalina Gallaron, for many favors and valuable assistance.

REFERENCES

(1) MANSON. Tropical Diseases. William Wood and Co., New York (1910).

(2) CASTELLANI and CHALMERS. Manual of Tropical Medicine. Baillière, Tindall and Cox, London. 2d ed. (1913).

(3) SAUNDBY and MILLER. *Brit. Med. Journ.* (1909), 1, 771.

(4) DAVIDSON. A System of Medicine. By many authors. Albutt, T. C., and Rolleston, H. D. Macmillan and Co., London (1907), **2**, pt. 2.

(5) CORLETTE. Amœbic dysentery and abscess of liver: An indigenous New South Wales case. *Med. Journ. Australia* (1915), **1**, 427.

(6) FUTCHER. A study of cases of amœbic dysentery occurring at the Johns Hopkins Hospital. *Journ. Am. Med. Assoc.* (1903), **41**, 480.

(7) TUTTLE. Cited by Anders and Rodman. The treatment of amebic dysentery, specially by appendicostomy. *Journ. Am. Med. Assoc.* (1910), **54**, 503.

(8) BOGGS. Amebic dysentery in the Southern States. *Virginia Med. Semi-monthly* (1908), **13**, 9.

(9) MUSGRAVE. Amœbiasis: Its association with other diseases, its complications and its after effects. *Phil. Journ. Sci.* (1906), **1**, 547.

(10) FERNANDEZ. Contribucion al diagnostico radiologico y al tratamiento medico quirurgico de los alcesos del higado. Actas, Memorias y Comunicaciones de la Segunda Asamblea Regional de Medicos y Farmaceuticos de Filipinas, Manila (1914).

(11) CROWELL. The chief intestinal lesions encountered in 1,000 consecutive autopsies in Manila. *Phil. Journ. Sci., Sec. B* (1914), **9**, 453.

(12) GILMAN. A report of the first one hundred autopsies at the Philippine Medical School. Ibid. (1908), **3**, 211.

(13) Cited by Davidson. No. 4.

(14) STRONG. *U. S. Army Med. Rep.* Washington, D. C. (1900).

(15) ROBINSON. Tropical abscess of the liver. *Trans. Nat. Assoc. U. S. Pension Exam. Surg.*, Rochester, N. Y. (1903), **1**, 161.

(16) MCDILL. Tropical infections of the derivatives of the primitive gut; their complication and treatment. *Surg., Gyn. and Obstet.* (1911), **13**, 523.

(17) Cited by Musgrave. No. 9.

(18) CRAIG. The etiology and pathology of amœbic infection of the intestine and liver. International clinics. J. B. Lippincott Com., Philadelphia and London (1905), **4** (XIV), 242.

(19) Cited by Craig. No. 18.

(20) Cited by Manson. No. 1.

(21) MUSGRAVE. Symptoms, diagnosis and prognosis of uncomplicated intestinal amebiasis in the tropics. *Journ. Am. Med. Assoc.* (1905), **45**, 830.

(22) ROGERS. Fevers in the Tropics (1910).

(23) WOOLLEY and MUSGRAVE. The pathology of intestinal amœbiasis. *Bull. P. I. Gov. Labs.* (1915), **No. 32.**

(24) Cited by Woolley and Musgrave. No. 23.

(25) COFFIN. Tropical liver abscess. Report of three cases with special reference to blood findings. *Am. Med.* (1906), **11**, 134.

(26) MCDILL. Dysenteric abscess of the liver in the Philippine Islands. *Journ. Am. Med. Assoc.* (1907), **49**, 491.

(27) ROGERS. Amœbic colitis in India: Prevalence, diagnosis and emetine cure. *Lancet* (1912), **2**, 1062.

(28) DEEKS and SHAW. Amebic colitis (amebic dysentery). *New Orleans Med. & Surg. Journ.* (1911), **64**, 1.

(29) CHALMERS and ARCHIBALD. The cure of amœbic dysentery. *Journ. Trop. Med. & Hyg.* (1915), **18**, 181.

(30) OSLER. Cited by McDill. No. 16.

(31) Dudley. Abscess of the liver rupturing into the lesser cavity of the peritoneum and giving rise to peculiar symptoms. *Journ. Am. Med. Assoc.* (1907), **49**, 39.

(32) Cyr. Quoted by Davidson. No. 4.

(33) Strong. Intestinal hemorrhage as a fatal complication in amœbic dysentery and its association with liver abscess. *Bull. P. I. Gov. Labs.* (1905), **No. 32.**

(34) Cited by Rogers. No. 22.

(35) Rhoads. Treatment of abscess of the liver. *Am. Med.* (1903), **6**, 659.

TWO CASES OF BALANTIDIAL COLITIS

By C. H. Manlove

(From the Department of Pathology, College of Medicine and Surgery, University of the Philippines)

The disease in man produced by *Balantidium coli* with a fatal termination can scarcely be called a common one in the Philippines, although, according to Walker,(4) "the presence of *Balantidium coli* in man in the Philippines is rather common."

An estimate of the occurrence of the disease in the Philippines may be taken from the following: Strong(3) recorded 1 death in 1904; Bowman(1) recorded 2 deaths up to 1911; Willets(5) recorded 2 cases in one of the provinces without a death; Walker(4) found 35 cases on the records at Bilibid Prison up to 1913, and he found 8 new cases in 1913; in the Philippine General Hospital there are 20 cases on record, including the 2 cases I wish to report. The number of deaths recorded in the Philippines is 5 from a total of 66 cases.

Strong, in 1904, reported a general review of 127 cases which he collected from the literature, including a case of his own. He found that of the 35 deaths recorded there were 32 autopsies performed, of which 28 showed an ulcerative colitis and 3 a chronic catarrhal colitis.

Walker worked on experimental balantidiasis, for which he infected monkeys with *Balantidium coli* derived from human beings and from pigs. Some of his conclusions are as follows:

> Parasitization of man with *Balantidium coli* is relatively common in the Philippine Islands. * * *
>
> A large proportion of the pigs in and about Manila are parasitized with balantidia. * * *
>
> Morphologically *Balantidium coli suis* is identical with *Balantidium coli hominis.* * * *
>
> The early lesions of the intestine of monkeys infected with *Balantidium coli* consist only of a slight hyperæmia with or without punctiform hæmorrhages.
>
> Histological examination of the tissues of monkeys recently infected with *Balantidium coli* shows changes, notably vascular dilation, minute hæmorrhages, round-cell infiltration and eosinophilia, which distinguish them from lesions of bacterial origin.
>
> *Balantidium coli* was never found entering the tissues through the lesions in 10 parasitized monkeys having a colitis or ulcerations due to bacteria or other causes.

In those monkeys in which infection took place, the balantidia entered the tissues through the sound intestinal epithelium.

Balantidium coli can produce bacteriologically sterile abscesses in the submucosa of an infected intestine.

Balantidium coli is the primary etiologic factor in the symptoms and lesions of balantidial dysentery.

The latency prevalent in balantidiasis of man is due chiefly to the fact that the patient, although parasitized, is not infected with *Balantidium coli*, but in part to the chronicity of the ulcerative process in infected cases.

Walker, in examination of the intestines in the early stages of the infection, found the epithelium intact except for a mechanical injury, which he attributed to entrance of the balantidia or to minute hemorrhages, but there was no exudate or ulceration. He found more or less congestion of the blood vessels, and the tissue infiltration, which was slight, was of round cells and eosinophiles. Those sections of the colon which did not show balantidia showed either a catarrhal or diphtheritic exudate or ulceration, usually associated with polymorphonuclear infiltration of the mucosa and submucosa.

The histological report of Strong is similar to that of Solowjew, which is as follows:

> The parasites were found invading the mucosa, submucosa and muscular layers. They were less numerous in the areas which showed marked necrosis and were found in the margins of more healthy tissue which surrounded these areas. The capillaries of the mucosa and submucosa were dilated, contained parasites and often showed hemorrhage about them. Often the amount of infiltration was small about single parasites in the tissues. However, when the parasites were found in large numbers the infiltration was very marked. The ulcerations extended in places down to the muscular coat, and their edges were usually undermined. The lymph glands were hypertrophied and hyperemic. No parasites were found in any other organ than the large intestine. In the mesenteric glands, spleen, liver, and kidneys there was considerable pigment inside the leukocytes in the blood vessels. No tubercle bacilli were found in the sections.

CASE REPORTS

CASE I

History.—The first case to be reported is one of a Filipino male, age 16, who was admitted to the Philippine General Hospital on March 1, 1916, at 3.30 in the afternoon, complaining of bloody diarrhœa, weakness, and loss of weight. Nothing could be determined as to his method of living or concerning the family and past history beyond the fact that he attended school.

The first intestinal symptoms began about two months previous to admission to hospital with frequent bloody bowel movements, which stopped after several days without treatment. Following this attack he had some irregularity of defecation with alternation of diarrhœa and constipation. During the month previous to admission there was a daily chill and fever, which continued until the present attack.

The present attack began about February 22, 1916, nine days before admission, with frequent bloody bowel movements which occurred almost every fifteen minutes. During this attack there was a moderate grade of fever, abdominal pain, and malaise.

He was treated at first for amœbic dysentery, and in an examination of the fæces, which was made before his admission to the Philippine General Hospital, no pathogenic organism was found. After his admission, before a laboratory examination had been made, a tentative diagnosis of bacillary dysentery, pulmonary tuberculosis, and malaria was made.

A summary of the physical findings is as follows:

The patient was restless, weak, and very much emaciated.

The pulse was small, weak, and rapid, and it fluctuated between 85 and 120, with an average of about 110 per minute.

The temperature varied between 36° and 38.5° C. It was generally subnormal in the early morning and might gradually or suddenly rise, sometimes varying as much as 3° C. in two hours.

The respiratory system showed areas of dullness and some crepitant râles over the apices. The respiratory rate averaged thirty per minute.

The nervous system was apparently normal, though the patient was dull and apathetic.

There was a more or less continuous diarrhœa throughout the course of the disease.

General course of treatment.—Throughout the eight days the patient was in the hospital, a continual proctoclysis of normal salt solution by the drop method was used. Proctoclysis of adrenalin chloride, 25 drops to 1 liter of salt solution, was also used. Quinine enemas of varying strength were used daily. On the second and third days after admission he received 30 cubic centimeters of normal horse serum intramuscularly, which produced a very appreciable effect in decreasing the amount of blood in the fæces.

Laboratory reports.—The number of bowel movements while in the hospital was 24, of which 15 occurred during the first three days. The fæces were thin, watery, blood-colored, and contained at all times large numbers of *Balantidium coli*, blood cells, mucus, and ova of *Trichuris*.

The urine was alkaline in reaction and free from sugar and contained a decided trace of albumin and abundant amorphous phosphates.

The blood was negative for typhoid and malaria. The hæmoglobin was 75 per cent. The leukocytes on March 2 numbered 8,400, while on March 3 they numbered 15,400, with a differential count as follows:

Polymorphonuclears, 85 per cent; small lymphocytes, 10 per cent; large lymphocytes, 1 per cent; transitionals, 2 per cent; and large mononuclears, 2 per cent.

The autopsy was performed March 9, 1916, at 9 o'clock in the morning, two and one-half hours after death. The fæces were examined at this time, and numerous motile *Balantidium coli* were found. The anatomic diagnosis was as follows: Acute and chronic ulcerative colitis (balantidial), intestinal hemorrhage, acute suppurative pleuritis, bronchopneumonia, acute parenchymatous nephritis, acute dilatation of the right ventricle of the heart, hyperplasia of spleen, trichuriasis.

The *body* is that of a very poorly nourished adult Filipino male, age 16 years, weighing 25.02 kilograms and having a length of 147 centimeters. The skin is pale brown and apparently normal. The eyes are sunken, and the corneæ are turbid. The hair is black, straight, and coarse and is limited to the pubes and scalp. The teeth are in fairly good condition. The superficial lymphatic glands are not palpable. Rigor mortis is present throughout the body, and suggillation is present in the dependent parts of the body.

On section the subcutaneous fat is almost completely absent, and the muscle has a brownish red color and is rather dry.

The *abdomen.* The serosa of the entire intestinal tract has a dark grayish color and is somewhat moist. The diaphragm is located at the fifth rib on the right and at the fifth interspace on the left. The abdominal viscera lie in normal relationship to each other. The omentum is free from fat.

The *thorax.* The thymus is very small, pale, and soft. On the right pleura are some fibrous adhesions posteriorly, but otherwise it is smooth and moist, and the sac contains no free fluid. The left pleural sac contains about 200 cubic centimeters of thin, creamy, and flocculent puslike material, which forms a thin coating over the lower lobe of the lung.

The *lungs* weigh 447 grams and float high in water, with the exception of the lower lobes, which are partially submerged. The upper lobes of both lungs crepitate throughout. The lower lobe of the left lung shows a large, consolidated area covering from 5 to 8 centimeters of lung tissue, which upon section shows a smooth grayish to brown surface with reddish brown, compact, raised, airless areas from which a very small amount of fluid exudes on pressure and which measure from 1 to 2 centimeters in diameter. The lower lobe of the right lung shows areas similar to those of the lower lobe of the left lung and also one large consolidated area of 4 to 5 centimeters in diameter. Many of these areas are abruptly outlined with apparently normal lung tissue. On section of the upper lobes they present a surface which is pale, smooth, air-containing, and apparently normal. The bronchi contain a thin mucoid material, and the mucosa shows no appreciable changes.

The *heart*. The parietal pericardium is pale gray, smooth, and thin. The pericardial sac is apparently normal. The epicardium is pale and smooth, though some of the coronary vessels have a tortuous appearance. The heart itself weighs 147 grams and is normal in shape. The auricles are filled with blood. The tricuspid ring almost admits four fingers, and the right ventricular cavity is enlarged and its walls are very thin. The mitral ring admits two fingers, and the ventricular cavity appears to be enlarged and the walls slightly thinner than normal. The muscle is dark brown and is very moist and rather firm. The blood is coagulated, forming coagula of chicken-fat consistence.

The *spleen* weighs 330 grams. It is firm and about normal in shape, and the capsule is pale gray. It cuts readily, showing a smooth, very compact dark brown surface, which is not very moist, and upon the surface the strands of interstitial tissue are visible.

The *adrenals* weigh 12 grams and are apparently normal.

The *kidneys* weigh 192 grams. They are about normal in shape and consistence. They cut easily, and the cut surface bulges. Upon the cut surface the pyramids are about normal in size, compact, and pale with reddened outline, while the cortex is pale with red linear marking. The capsule strips very easily, exposing a pale surface marked with numerous stellate injections.

The contents of the *small intestine* vary from a thick, creamy yellowish brown material in the jejunum to a thick blackish fluid in the lower ileum. The contents of the colon vary from a thick black fluid in the ascending and transverse colon to a soft, semisolid, thick, turbid black fluid in the descending colon. Trichurides are present.

The walls of the *colon* are somewhat thickened and are not friable. The mucosa is more or less ulcerated throughout its entire length. In the rectum and adjoining 5 to 10 centimeters of the descending colon it shows the most ulceration, and here the mucosa is reddened, thickened, and riddled with ulcers. In the cæcum it shows almost as numerous ulcers, but here they are more extensive than in the rectum. In the transverse colon and other parts of the colon the ulcers are scattered more widely apart.

The ulcers may be divided into three stages according to the degree of involvement of the colon wall: an early, medium, and late. All three stages are found throughout the entire colon, but predominate as follows: Those in the cæcum are mostly in a late stage with some early and medium found here and there; those of the rectum are mostly of the medium stage with the late stage next in frequency; those of the transverse colon are of all three stages, of which the medium predominates, with one very large late ulcer measuring 4 to 5 centimeters in diameter, which has very irregular, undermined, reddened edges, with a flat base upon which the muscle is visible. The early ulcer is evidenced by a slight, firm elevation of the mucosa, which is reddened and shows a pale central depressed area, which measures from pin point to 1 or 2 millimeters. The medium ulcer is evidenced by a distinct, slightly raised, hemispherical area with a depressed, necrotic center, which measures from 1 millimeter to 1 centimeter. The edges are puckered, irregular, undermined, and deeply reddened and have a reddened areola of mucosa about them. The bases are pale and necrotic, appear flat, and are located in the submucosa. The late ulcers average about 2.5 centimeters in diameter. They are very irregular in shape, with edges that are not elevated but are deeply undermined. The bases are flat and smooth, are located on the muscular coat, and are covered with necrotic material.

The mucosa of the *small intestine* is pale, smooth, and moist and is apparently normal.

The *stomach* is normal.

The *pancreas* is pale and firm, but shows no other changes.

The *mesentery* is practically free from fat, and the lymphatic glands are very pale, but apparently normal.

The *liver* weighs 1,235 grams. It retains its shape after removal and is slightly firmer than normal. The capsule is smooth, glistening, and bluish brown. The liver cuts easily, showing a smooth dark brown surface which is rather moist; the lobules are not distinct. The bile ducts and vessels are apparently normal.

The *gall bladder* contains a very thick, viscid black bile, while the mucosa and walls are apparently normal.

The *urinary bladder* contains a small amount of amber-colored urine, and the mucosa is apparently normal.

Histologically the colon presents changes which vary from a simple catarrh to extensive ulceration involving the muscular coat.

Changes in the mucosa.—The mucosa of all parts examined has undergone more or less catarrhal inflammation, and in places it is covered with a superficial pseudomembrane of hemorrhagic and necrotic material. The capillaries are dilated, and many of them are ruptured.

Here and there may be seen distinct ulcers of 1 to 2 millimeters in diameter whose base is superficial to the muscularis mucosæ. There are also areas of 2 to 3 millimeters in diameter where the superficial epithelium has desquamated, while the epithelium at the base of the glands and the interglandular papillæ remain. The papillæ are necrotic near the surface, but in the deeper parts they are thickened, fibrous, and œdematous and are infiltrated with plasma cells and lymphocytes. The blood vessels in the superficial parts of the papillæ are widely dilated and ruptured and communicate with the surface, which is covered with a pseudomembrane containing balantidia.

Where the mucosa forms the margins of the ulcers, there is formation of goblet cells, desquamation of epithelium, necrosis, and infiltration of plasma cells, of round cells, and of some polymorphonuclear neutrophile leucocytes. The blood vessels are dilated, ruptured, and partially or completely obliterated with fibrin thrombi, and in many instances they communicate with vessels in the submucosa showing similar changes.

The balantidia in relation to the mucosa are found on the surface, in the tubules of the glands, within the glandular walls, in apposition to the muscularis mucosa, just penetrating the glandular basement membrane, in the superficial ulcers, and in the solitary lymphoid follicles. They are found both in direct relation to ulcers and in mucosa which is not in close contact with ulcers. In no instance is there any definite cellular reaction surrounding a singly located organism.

Changes in the submucosa.—The ulceration in places extends to the submucosa, in which the change varies in degree from a superficial involvement to one extending to the muscle. The ulcers have overhanging, undermined edges which curl into them. They are deeper at their lateral extremities, leaving a raised necrotic base whose apex in some instances is level with the surrounding mucosa, giving the ulcer an inverted bowl shape and leaving considerable submucosa in apposition to the undermined mucosa. Surrounding the ulcers the submucosa is œdematous, thickened, and more or less infiltrated with round cells and polymorphonuclear neutrophiles in groups and more scattered. The infiltration is closely limited to the ulcer and is least in evidence beneath the intact mucosa beyond the margins. The margins of the ulcer are densely infiltrated and show considerable necrosis. Here the blood vessels are widely dilated, some being ruptured and many partially or completely obliterated with fibrin thrombi.

The openings of the ulcers through the mucosa vary from wide openings of 1 to 2 centimeters to fistulous openings of 1 to 2 millimeters in diameter, and these communicate with large cavities in the submucosa which are filled with balantidia and necrotic material. Some of the superficial ulcers involve the solitary lymphoid follicles. Balantidia are found scattered through these follicles, and also there may be groups of twenty or more balantidia found in the lateral deep margins of the ulcers.

The balantidia invading the deeper part of the submucosa are found at the lateral extremities of the ulcers in groups of fifty to a hundred, where there is usually much necrotic material about them and much peripheral cellular infiltration. Scattered through the base and intermingled with the necrotic contents of the ulcer, they are in small numbers and usually singly. They are also present in the tissues about the ulcers, singly and in groups, where they are surrounded by a dense cellular infiltration. They may be seen within the blood vessels of the margins and bases and elsewhere in the submucosa in close relation to the ulcers. The balantidia occur in large and small groups where the submucosa is intact about them, although in this case they are in close relation to an ulcer or the mucosa over them shows inflammatory changes. Here there may be much or little cellular infiltration about them. Those found in vessels or singly are not surrounded by any cellular infiltration, while those in groups are surrounded by more or less cellular infiltration.

Changes in the muscle.—The muscle is involved by, and forms the base for, the most extensive ulcers, at which places it is infiltrated with plasma cells, round cells, and polymorphonuclear neutrophiles in groups and scattered and shows some necrosis. The blood vessels about the involved areas are widely dilated and surrounded by cellular infiltration. The subserous tissue in apposition to the ulcers is œdematous and slightly infiltrated, while the serosa opposite the extensive ulcers is covered with a fibrinohemorrhagic material, which shows some cellular infiltration.

Balantidia are found within blood vessels of the muscle, also in the subserous tissue, where they are both within blood vessels and lying free. They are found in varying numbers in the necrotic material covering the bases of the ulcers, where there is cellular infiltration about them, while the organisms found in the muscle and subserous tissue are free from any surrounding cellular infiltration.

Summary of first case.—A 16-year-old Filipino was in hospital for seven and a half days before death. He had bloody diarrhœa for a few days two months before death, followed by intermittent diarrhœa and constipation. There was a daily chill and fever during the last month. For sixteen days before death there were frequent bloody diarrhœa, moderate fever, abdominal pain, and malaise. Physical examination one week before death revealed emaciation, weakness, slight tachycardia, changes in temperature varying from 36° to 38.5° C., albuminuria, and slight leucocytosis. The fæces were thin, watery, and blood-stained and contained at all times large numbers of balantidia, blood cells, mucus, and ova of *Trichuris.* Autopsy revealed a colitis with bronchopneumonia and degenerative lesions of the viscera.

CASE II

History.—The second case was a Filipina, age 51 years, who was first admitted to the hospital August 23, 1916, and a diagnosis was made of mixed tumor of the parotid and epithelioma of the cheek. She was discharged September 1, 1916, as incurable. At this time there was no record of intestinal trouble. The patient was again admitted to the hospital September 21, 1916, complaining that one week previously she had become too weak to walk. She was very poorly nourished, with little or no appetite, and was constipated. On the forehead were three small superficial lacerated wounds. On the left side of the neck just below the ear was an irregular swelling with a sinus about 1 centimeter in diameter discharging a moderate amount of pus. The left corner of the mouth was indurated, and this induration was continuous with an induration and ulceration on the inner surface of the cheek, the tongue, and the gums in the region of the lower molars. On the right forearm just above the wrist was an ulcer at the base of which the ulna was exposed. On the buttocks there were numerous scars and one ulcer with undermined edges. During the ten days after admission, from September 21 until October 1, the bowels remained constipated, and there was no change in the general condition, although the external wounds had healed. However, from October 1 to October 4 there was slight diarrhœa, and from October 4 to October 13 there was an alternation of constipation and slight diarrhœa. During this time the bowels would be constipated for one or two days without any evacuation, which would be followed by several evacuations the next day. She became progressively weaker and died October 13, 1916.

The temperature during the twenty-four days in the hospital remained subnormal, varying from 35.5° to 36.5° C. On two occasions it rose to 38° C. within four hours and as rapidly became subnormal. The pulse fluctuated between 80 and 90 per minute.

The respiration averaged 20 per minute. The number of stools varied from one to three a day with nine days without any passage of stools.

Laboratory reports.—The fæces on September 27, 1916, showed *Balantidium coli*, encysted amœbæ, ova of *Trichuris* and *Ascaris*, blood, and pus. On October 4 they showed numerous motile *Balantidium coli* and some *Blastocystis hominis*. On October 8 there were no balantidia. The urine showed a decided trace of albumin, some pus and red blood cells, granular casts, epithelial cells, and mucus. The blood was not examined.

The clinical diagnosis was: Epithelioma of mouth, involving cheek, tongue, and upper jaw, mixed tumor of parotid, balantidiasis, trichuriasis, ascariasis, amœbiasis (intestinal), blastocystiasis, and inanition.

The autopsy was performed October 14, 1916, fourteen hours after death. An examination of the fæces at this time failed to reveal any balantidia. The anatomic diagnosis was: Chronic catarrhal colitis (possibly balantidial), chronic diffuse nephritis, chronic ulcerative tuberculosis of the lung, infected tumor of parotid, ascariasis, and trichuriasis.

The *body* is that of a very poorly nourished adult Filipina, whose age is 51 years, weight 25.10 kilograms, and length 157 centimeters. The skin is dark brown and is shrunken over most of the body. On the lateral surface of the right hip there are several open ulcerative lesions about 2 centimeters in diameter. These extend into the subcutaneous tissue. On the right forearms there are ulcerative lesions, which extend into the subcutaneous tissue. The corneæ are very turbid, and the eyes are sunken. The mucosa of the mouth is covered with a whitish necrotic material. Many of the teeth are missing, and those present show extensive caries. At the left posterior angle of the jaw there is a raised mass which measures about 7 centimeters in diameter and is elevated about 3 centimeters above the surrounding tissue. This shows two sinuses, which penetrate into the mass, and from them exudes a thick, creamy, puslike greenish material. The mass involves the region of the left parotid and submaxillary gland. The central part of it is necrotic and is filled with a greenish puslike material, and the cut surface is compact, whitish, and firm. Rigor mortis is present to some extent throughout the body, and suggillation is present in the dependent portions of the body.

On section there is practically no subcutaneous fat. The muscle is light reddish brown and moist.

The *abdomen.* The serosa of the entire intestinal tract, parietes, and stomach is pale, moist, and apparently normal. The diaphragm is normally located. All the abdominal viscera have gravitated somewhat toward the pelvis.

The *thorax.* The thymus is almost completely absent, and fatty tissue has taken its place.

The *pleural sacs.* The left pleural sac contains fibrous adhesions in the apex, and the right pleural sac is free from adhesions. Otherwise both sacs are moist, do not contain free fluid, and appear normal.

The *lungs* weigh 500 grams. They float very high in water and are large, and crepitation is present throughout, except in the apex of the left lung, where there is a consolidated area of about 5 to 6 centimeters in diameter. Section through this lung shows an abscess cavity of 3 to 4 centimeters in diameter, which contains a thick, creamy grayish yellow pus and whose walls are rough and necrotic. The lung tissue immediately about the abscess is compact and whitish and contains caseous material. Section through the lungs elsewhere shows a smooth, moist reddish surface which is air-containing and apparently normal. The pleura covering the lungs is pale gray with black pigmented mottling. The bronchi have a grayish color, and those of the left lung contain some puslike material.

The *heart.* The pericardium and pericardial sac appear normal. The epicardium has a grayish color, and the coronary vessels are tortuous. The heart itself weighs 176 grams. It is rather firmly contracted. The tricuspid ring admits almost three fingers, and the mitral ring admits barely two fingers. The endocardium covering the valves is thickened, firm, and somewhat roughened. This is noticeable at the bases and the edges of the leaflets. The ventricles and auricles show no appreciable change from normal. The muscle is brown, and its cut surface is smooth, dull, and moist. The blood is coagulated, forming a coagulum of chicken-fat consistence.

The *spleen* weighs 37 grams. It is about normal in shape and somewhat soft in consistence. The capsule is blue. The spleen cuts easily, showing a smooth, moist, compact reddish surface.

The *adrenals* weigh 9 grams. They are rather firm in consistence, dark brown in color, and show no appreciable change.

The *kidneys* weigh 217 grams. The left kidney measures 12.5 centimeters in length by 6 centimeters in width and 3 centimeters in thickness. The right kidney measures 9 centimeters in length, 5 centimeters in width, and 3 centimeters in thickness. Both kidneys are somewhat firm in consistence and about normal in shape. They cut readily, showing a smooth, moist surface, from which a small amount of blood exudes. Upon the surface the pyramids are very large, compact, glistening, and reddish, while the cortex is mottled and light brown and red, although in places the columns of Bertini are pale and bulging. The capsule strips readily, showing a surface which is slightly roughened by depressions of 0.5 centimeter in diameter, and it has numerous stellate injections upon a light brownish background. In places it is firmly adherent to the kidney substance.

The *intestines*. The serosa has been described. The contents of the small intestine are a thin, mucoid yellowish and brown material, while in the colon the contents vary from a thick, semifluid brownish material in the ascending and transverse colon to a thick, mucoid grayish material in the descending colon. The mucosa throughout is thickened, swollen, soft, and velvety, and it has a dark grayish color with a reddish tinge in places, although in the descending colon the mucosa is mottled with what appear to be superficial ulcers. These areas measure about 3 or 4 mil-

fluid brownish material in the ascending and transverse colon to a thick, mucoid grayish material in the descending colon. The

base appears to be located in the mucosa. The mucosa of the small intestine is pale grayish, smooth, and moist and is apparently normal.

The *stomach* contains some thick mucoid material. The mucosa is grayish and smooth.

The *pancreas* is firm in consistence, but normal in size and shape and shows no appreciable change.

The *mesentery* appears normal.

The *liver* weighs 956 grams. It is reddish brown and is about normal in shape and consistence. The capsule is mottled in places. The liver cuts readily, showing a smooth, moist, glistening surface, which has a reddish brown mottled appearance. The lobules are plainly visible and show a reddish center and a pale periphery. The bile ducts are surrounded in places with considerable fibrous tissue, and they have a yellowish tinge throughout. The common bile duct measures about 1 centimeter in diameter and contains four or five dead ascarides.

The *gall bladder* was removed intact for bacteriological examination.

Histologically the changes in the colon are confined to the mucosa. The mucosa shows catarrhal inflammation and some desquamation of the superficial epithelium, and the tubules are filled with mucus. The mucosa is frequently folded in such a manner that the shape of an ulcer is produced with undermined edges. These areas are completely lined with epithelium, but may contain fæcal material within which balantidia are embedded. The balantidia are found only on the surface of the mucosa, where they are embedded in fæcal material. The tumor in the mouth proved to be an epithelioma.

Summary of second case.—A 51-year-old Filipina was under observation about seven weeks. She sought hospital treatment on account of epithelioma of the cheek and a mixed parotid tumor. Weakness was a marked symptom. She was constipated until ten days before death, when intermittent attacks of diarrhœa occurred. She was emaciated and had no appetite, and there were subnormal temperature, normal pulse, normal respiration, and albuminuria. The intestinal condition only assumed importance after examination of the fæces and the finding of balantidia. Autopsy showed a colitis as a complication of the other more important disease.

DISCUSSION

In the first case I was particularly careful to exclude the possibility of balantidiasis of the left lung and pleural sac. It is of interest to note that cultures made from the pleural sac remained sterile. The histological examination of the lung and pus did not reveal any balantidia. In view of the fact that the sections of colon show balantidia in blood vessels, there must be a continual entrance of balantidia into the general circulation. However, it is rare to find them producing lesions outside of the intestines, although they have been reported in sputum from a case of supposed lung involvement. In my cases the physical findings cannot be said to be distinctive of balantidiasis. The temperature was one of the most variable features, as it fluctuated from subnormal to above normal. The absence of eosinophilia in both of these cases seems to support the belief that eosinophiles may or may not be found in all intestinal disturbances. Since the balantidia are found penetrating both the mucosa and submucosa without any surrounding cellular infiltration, it may be safely concluded that they are capable of penetrating apparently healthy mucosa. Here they may produce sterile abscesses which may in time become secondarily infected by the rupture of the mucosa covering them and thus form ulcers. Macroscopically the lesions produced by *Balantidium coli* cannot be differentiated from those of *Entamœba histolytica.* Microscopically the lesions show a similar cellular reaction, but are easily differentiated by finding the organisms. Many of the balantidia examined in the wall of the colon did not show the typical nuclei, but contained numerous small masses of nuclear-staining material, which resemble chromidia formation, while those examined on the surface of the mucosa show the typical nuclear structures.

As suggested by Dr. B. C. Crowell, the question of the essential pathogenicity of balantidia is an important one, and one which cannot yet be considered to be absolutely settled. It is necessary to determine the invasive powers of the balantidia, that is, whether they may or may not of themselves penetrate the intact mucosa, and if so, whether they pass through the epithelial cells or between the cells. Reasoning from analogy with known facts concerning the action of entamœbæ, and from the known tissue changes described in balantidiasis, it seems necessary to believe that these organisms are essentially pathogenic and that their action is in all ways similar to the action of the entamœbæ. Doctor Crowell expressed the theory that it seems reasonable to conclude that some substance (toxin?) originating from the balantidia is able to produce necrosis of the tissues, and the inflammatory phenomena that occur later are the result of the action of the accompanying bacteria. Also that one could imagine that this hypothetical toxic substance from the balantidia produces a cytolysis of the epithelial cells of the mucosa with which they come in contact and so forms a portal of entrance to the deeper tissues for the organisms.

SUMMARY

1. Two cases of balantidiasis with clinical histories and autopsy reports have been added to the five previously published autopsy cases from the Philippines.
2. In one case there was a history of dysentery for approximately two months before death; in the other the intestinal

condition was detected only on routine examination and was unimportant from the clinical standpoint.

3. Balantidiasis is relatively unimportant as a cause of death in the Philippines, but may cause considerable morbidity.

4. As in amœbiasis, extensive intestinal lesions in balantidiasis may be present without giving rise to symptoms.

5. The intestinal lesions in balantidiasis vary from a simple catarrh to deep ulceration. They are characterized by abundant organisms in the intestinal wall with some inflammatory manifestation and by a burrowing necrosis of the submucosa, which is similar to the amœbic lesion. All parts of the colon may be involved. An etiologic diagnosis can only be made by finding the protozoa. Eosinophilia in sections of the intestine has not been prominent in these cases, although it has been emphasized by previous authors.

REFERENCES

(1) BOWMAN, F. B. A case of dysentery caused by Balantidium coli with coincident filarial infarction of the spleen. *Phil. Journ. Sci., Sec. B* (1911), **6**, 147–152.

IDEM. Two cases of Balantidium coli infection. *Phil. Journ. Sci., Sec. B* (1909), **4**, 417–423.

(2) SOLOWJEW, N. Das Balantidium coli als Erreger chronischer Durchfälle. *Centralbl. f. Bakt. etc., 1. Abt.* (1901), **29**, 821, 849. Cited by Strong (3).

(3) STRONG, R. P. The clinical and pathological significance of Balantidium coli. *Pub. Bur. Govt. Labs.*, Manila (1904), **No. 26**, 1–77.

(4) WALKER, ERNEST LINWOOD. Experimental balantidiasis. *Phil. Journ. Sci., Sec. B* (1913), **8**, 333–351.

(5) WILLETS, D. G. General conditions affecting the public health and diseases prevalent in the Batanes Islands, P. I. *Phil. Journ. Sci., Sec. B* (1913), **8**, 49–58.

Issued with THE PHILIPPINE JOURNAL OF SCIENCE, SECTION B,
Tropical Medicine, Vol. XII, No. 3, May, 1917.

PROCEEDINGS OF THE MANILA MEDICAL SOCIETY

REGULAR MONTHLY MEETING, MARCH 5, 1917

The regular monthly meeting of the Manila Medical Society was held at 8.30 in the evening, March 5, 1917, in the College of Medicine and Surgery. President Winter was in the chair; 19 members and 2 guests were present.

The minutes of the last meeting were read and approved as read.

As there was no further business to come before the society, the president announced the first paper for the evening, namely, by Dr. José Hilario and Mr. L. D. Wharton on the anatomy of *Echinostoma ilocanum*, with clinical report on some recent cases. The paper was followed by an excellent demonstration of the characteristic anatomy of the Echinostoma.

Dr. José Albert then presented an interesting paper on his experience in the diagnosis and treatment of infantile beriberi. He described an acute, a chronic, and a pseudomeningitic form and described the last form in detail.

The president then asked Doctor Arvisu to read the clinical records of the cases of pseudomeningitic beriberi described by Doctor Albert.

Doctor Gibson next gave a demonstration of the Folin methods for the examination of urine. By these methods he was able to demonstrate that the amount of nitrogen, ammonia, and urea in urine could be determined in approximately thirty-five minutes.

It was then moved and seconded that the society adjourn.

H. G. MAUL,
Secretary-Treasurer,
Manila Medical Society.

SCIENTIFIC PROGRAM

ECHINOSTOMA ILOCANUM (GARRISON): A REPORT OF FIVE CASES AND A CONTRIBUTION TO THE ANATOMY OF THE FLUKE

By Dr. J. S. HILARIO and Mr. LAWRENCE D. WHARTON

Five cases of infection with *Echinostoma ilocanum* are reported in patients at the Philippine General Hospital. All of the infections were in natives of Zambales Province, Luzon. The symptoms presented were anæmia, with occasional headache and dizziness. The blood picture showed more or less marked decrease of the hæmoglobin percentage, moderate diminution in number of red corpuscles, and eosinophilia in some cases. Treatment consisted of 24 capsules of oleoresin of male fern given in doses of 4 capsules every ten minutes. During the previous day the patient was kept on liquid diet without milk as a preparatory measure for the treatment.

Sixteen worms were obtained from two of the cases after treatment. Specimens ranged from 4.03 millimeters to 7.82 millimeters in length and from 0.98 millimeter to 1.6 millimeters in breadth. The cuticular spines were found to be absent in the majority of specimens and were very easily broken off in those specimens where they were present. Only three specimens showed the oral wreath of spines which is characteristic of the genus. In the one specimen on which these spines could be counted they were found to number 39. The arrangement was similar to that described by Odhner. The structure of the internal organs was found to agree very closely with Garrison's description. Measurements of 25 fresh eggs from fæces gave an average of 101.2 microns by 56.4 microns.

This paper will appear in full with illustrations in a future number of the Philippine Journal of Science.

THE PSEUDOMENINGITIC FORM OF INFANTILE BERIBERI

By Dr. JOSE ALBERT

In the diagnosis of infantile beriberi it may be rightly said that the medical man continues to be a student as long as he remains in the profession. Fifteen years ago, when beriberi in infants under 1 year of age was still unknown, I wrote in the death certificates of such cases diagnoses of convulsions, heart failure, pneumonia, and peritonitis. In the twelve years since the existence of infantile beriberi as a distinct disease has been confirmed and generally recognized, two clinical forms or types of this important disease are admitted: the chronic aphonic type which lasts for several weeks and in which hoarseness of voice associated with hypertrophy and dilatation of right heart are the predominant symptoms, and the acute, pernicious, cardialgic type, lasting from several hours to two days, and characterized by intense and incessant crying.

During last year, 1916, I had the opportunity to observe three cases of infantile beriberi entirely distinct from the two types described above. They were characterized by cerebromeningeal symptoms. I have applied the name "pseudomeningitic form" to this type of the disease. As far as I know, this form has never been described in this country.

The three cases I referred to were treated unsuccessfully outside our clinic as cases of meningitis. Their age varies from 5 to 6 months, somewhat older than the ordinary cases. All were entirely breast-fed, and the mothers had symptoms of rudimentary or latent beriberi. Two of the cases had brothers and sisters who died of infantile beriberi, while the third case was the first child of the family.

The most striking symptom in these patients was drooping of the eyelids—ptosis—a paralytic manifestation of the neuromuscular form according to Professor Takasu, of Osaka. Because of the paralysis of the eyelids, the child has a vacant, idiotic expression. He is drowsy most of the time, the eyes looking upward and the head bent slightly backward. Moderate fever was present at the onset in two cases, while absent in the third. One had marked choreic movements of the limbs, which lasted for from three to four weeks, and on admission this patient presented convulsions of the extremities, twitching of the facial muscles, strabismus, the eyes rolling up, slight rigidity of the neck, and fever of 38.5°C. for two days. Lumbar puncture was performed on this patient with negative results. Two gave a history of having hoarseness of voice at the age of two months, which fact suggests that these cerebromeningeal symptoms are manifestations of a second attack of infantile beriberi.

In all these cases X-ray examinations demonstrated the enlargement of the right heart, the characteristic lesion of the disease.

All were treated with extract of tiqui-tiqui with rapid and marvelous effects, as observed in other forms of infantile beriberi.

In view of these observations I feel justified in presenting to your consideration this pseudomeningitic form of infantile beriberi as a new type of the disease distinct from the aphonic and pernicious types. This form of infantile beriberi simulates meningitis in its clinical manifestation; it presents hypertrophy and dilatation of the heart and is observed in infants older than 3 months, and extract of tiqui-tiqui is its specific remedy.

A DEMONSTRATION OF THE FOLIN METHODS FOR THE ANALYSIS OF THE URINE

By Dr. R. B. Gibson

The publication by Folin and Denis[1] of a description of practical colorimetric methods for the determination of the several forms of nitrogen in the urine and blood makes a demonstration of these methods timely. The methods, which are based on direct Nesslerization, are easy, rapid, and accurate. With a little practice they may be carried out by the ordinary physician. These methods are at present being taught to the students in physiological chemistry in the College of Medicine and Surgery of the University of the Philippines. Printed directions for the analyses, as demonstrated before the society, will be furnished on application to the department of physiology.

R. B. Gibson,
Editor of the Proceedings,
Manila Medical Society.

---

[1] *Journ. Biol. Chem.* (1916), 26, 473–506.